ART. III.—THE ATHEROMATOUS PROCESS IN ITS RELATIONS TO THE BRAIN.

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I.—PATHOLOGICAL ANATOMY.

THE atheromatous process consists of a parenchymatous inflammation of a connective tissue membrane, usually the *intima*, or lining membrane of the arteries, which becomes thickened, and either gelatinous or cartilaginous in structure, by proliferation of the connective tissue in the form of flattened gray or yellowish elevations.

In the former case the superficial, otherwise the deepest layer of the intima, subsequently becomes the seat of fatty metamorphosis of these inflammatory products in the cells of the connective tissue lamellæ, thus giving rise, after the destruction of the cell membranes, to a semi-fluid mass, consisting of fat granules, calcium salts, crystals of cholesterine, and amorphous fragments, forming the *atheromatous abscess*. If the inner lamellæ covering the abscess is destroyed, we obtain the atheromatous ulcer. The latter, being in contact with the blood, gives rise to coagula of fibrine, on account of its uneven edges, while particles from the ulcer are carried away by the current of blood, and may thus lead to obstruction of vessels. Finally, calcification occurs by the deposition of calcium salts, or even ossification by the formation of plates of bone in the lamellæ of the connective tissue. The osseous plates extend into the interior of the vessels, and thus afford opportunity for the formation of clots. In consequence of the atheromatous process, the arteries dilate on account of the degeneration and atrophy of their muscular coat, while the smaller arteries become narrowed and even obliterated from the thickening, which generally extends also to the middle coat. The smallest arteries form miliary aneurisms, caused by the atony of their tunics and the degeneration of the middle coat, the vessel being thus enlarged by the blood-pressure.

obliterated vessel. These occlusions can be occasioned by other products as well as the depositions of fibrine. Thus, embolisms may consist also of necrosed fragments of the atheromatous walls of vessels, pigmentary agglutinations, etc., and not rarely do we find thrombi formed of the products of degenerated blood-corpuscles, as may be proven by the anatomical characters and the presence of pigment. Though the disturbances of nutrition caused by the occlusion are the same as those resulting from the atheroma itself, I still wish to point out this difference, that in the former cases the textural changes are partial, and occur at a time before the atheroma itself can, as yet, produce the same in the whole brain.

Another consequence of the atheromatous process is, finally —as we have already shown for the smaller arteries—the dilatation, either circumscribed or involving the vessel to some distance—the ordinary aneurism; or when the atheromatous inner and middle coats have burst, and the blood dissects off the adventitia, which finally ruptures also—the dissecting aneurism. As another explanation for the occurrence of some aneurisms, Ogle * found in his experiments, that in occlusion of a vessel (embolism or thrombosis), the plug of fibrine is wedged in tighter by the *vis a tergo* of the blood, while the elastic walls of the vessel become distended by the lateral pressure of the blood; hereupon they lose their contractility, especially when they are atheromatous or otherwise diseased; and when the surrounding tissues are very yielding, as in the brain, an aneurism is formed. The effects of an aneurism are like those of a cerebral tumor; very variable with the size and extent, as well as the locality of the same.

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* John W. Ogle, *On the formation of aneurisms in connection with embolism or arterial thrombosis.* London: 1865. Benj. Pardon, pp. 8.